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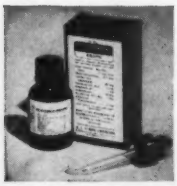

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WRINGER INJURIES IN CHILDHOOD

Special Report No. 188

Charles Webb, M. D.
Joseph M. LoPresti, M. D.

This report represents a survey of children seen at Children's Hospital in the past two years who sustained accidental washing machine wringer injuries. An extensive review reveals that similar reports are singularly absent in the medical literature. Since such injuries occur with relative frequency in the pediatric age group, it was felt that an investigation into this subject would be of interest to the medical practitioner. It seems to be the general consensus of opinion that such injuries are rarely attended by serious complications other than some minimal soft tissue damage, and that fractures are rarely, if ever, encountered. Our review, however, would indicate that this concept is erroneous. This report was prepared with the aim of acquainting the physician with the complications that may arise in a child who has caught his arm in a wringer. On occasion, the damage may be so severe as to require long periods of hospitalization.

In the past two years, forty-three children who sustained wringer injuries were examined. The age, sex, and race distribution of these cases are illustrated in Table I. There were twenty-seven males and sixteen females. Seventeen of these children were white and twenty-six were colored. Their ages ranged from one to ten years with an average of three years, ten months. Thirty-five (81.4 per cent) of these children were between the ages of one and five years; only eight were over six years old.

Complications occurred in ten (23 per cent) of these patients. These are summarized in Table II. The complications are:

1. *Soft Tissue Damage.* The soft tissue damage which may follow this traumatic incident varies from moderate edema to extensive necrosis. The necrosis may extend to all of the layers of the epidermis and dermis, and in rare instances, may involve even the underlying muscles and tendons. When severe involvement occurs, it may be necessary to utilize surgical grafting to prevent scar formation and deforming contractures. An occasional patient will develop a fluctuant abscess in the region of the injured area which will ultimately require surgical incision and drainage. Soft tissue damage occurred in five (11.3 per cent) of the children. One of these required skin grafting (cf. Case Report).

2. *Fractures.* The consensus seems to be that fractures rarely, if ever, are encountered in this type of trauma. However, four children (9.3 per cent) sustained fractures of one or both bones of the forearm. In two patients

both the radius and ulna were fractured. In the remaining two only the ulna was involved. While the incidence of fractures is small, they do occur in sufficient number to make it incumbent upon the physician to have all

TABLE I

The Age, Sex, and Race Distribution of 43 Children Who Sustained Wringer Injuries

AGE	NO. OF CASES
0– 1 year	0
1– 2 years	11
2– 3 years	11
3– 4 years	6
4– 5 years	7
5– 6 years	4
6– 7 years	1
7– 8 years	0
8– 9 years	1
9–10 years	2
Total	43

Males	27	White	17
Females	16	Colored	26
Total	43	Total	43

TABLE II

The Complications Which Occurred in 43 Children Who Sustained Wringer Injuries

COMPLICATION	NUMBER OF CASES
I. Soft Tissue Damage	5
A. Moderate edema	2
B. Abscess of injured area	1
C. Extensive tissue necrosis	2
II. Fractures	4
A. Radius and ulna	2
B. Ulna alone	2
III. Localized osteomyelitis	1

wringer injuries examined by x-ray. The medico-legal implications are clear. All of the children in this survey had roentgenographic examinations of the injured part.

3. *Osteomyelitis.* The soft tissue damage and necrosis which may accrue from a wringer injury are ideal foci for bacterial contamination. When such

bacterial invasion occurs, it may extend to the underlying bones and produce a localized osteomyelitis. Bones, so weakened by this inflammatory reaction, provide excellent sites for pathological fractures. This sequence of events occurred in one of the patients in this series (cf. Case Report).

FIG. 1. X-ray of the right forearm which reveals the pathological fracture of the ulna. Note the osteosclerosis and periosteal elevations in the region of the mid-shafts of both bones which were interpreted as healing osteomyelitis.

CASE REPORT

The following case report serves to illustrate the more serious sequelae of a wringer injury.

B. S., a six year old white female, was admitted to the hospital on April 21, 1949. The history revealed that she had caught her right forearm in a washing machine wringer on the afternoon of admission. The parents had returned home and found her with her right arm in the wringer. The time interval during which the arm was trapped was unknown. Physical examination was normal except for the right upper extremity. There was considerable edema extending from the elbow to the wrist. Two deep abrasions, 7 by 5 centimeters each, were located on the ventral and dorsal surfaces of

the arm respectively, just below the elbow joint. The large blood vessels were exposed in both of these areas. In addition, numerous hematomata and bruises were scattered over the right forearm.

Immediate therapy included débridement and cleansing of the open wounds, parenteral penicillin prophylactically, and continuous hot saline soaks to the right forearm.

An x-ray of the right upper extremity on admission revealed no evidence of fracture or any other osteal or periosteal pathology.

One week after admission a paravertebral block was performed at the level of the second and the third thoracic vertebrae in the hope that this measure would improve the circulation to the injured part.

The next six week period of hospitalization was spent in preparing the patient for skin grafting and in preventing contracture deformities with physiotherapeutic measures. Finally an abdominal flap was attached to the arm with good results. The donor site healed without complication. However the recipient site became infected and continued to drain a small amount of purulent material for the next four week interval. Culture of this wound yielded *Hemolytic Staphylococcus aureus* and *Aerobacter aerogenes*. These organisms were found to be extremely sensitive to aureomycin. Penicillin therapy was discontinued and treatment with aureomycin, 500 milligrams every eight hours by mouth, was instituted. At no time during hospitalization was a sustained febrile period noted.

On September 6, 1949, approximately five months after admission, an x-ray of the right forearm revealed a pathological fracture of the right ulna at the junction of the distal and middle thirds. Some osteosclerosis of the mid-shaft of the radius and ulna was noted. This was interpreted as representing a healing osteomyelitis (Figure 1). In spite of intensive aureomycin and penicillin therapy the pathology in the right forearm progressed to involucrum formation. The patient was finally discharged from the hospital on November 9, 1949 after a period of hospitalization which lasted approximately 7 months.

She has since been followed in the orthopedic clinic. At the present time atrophic changes are evident in the distal portion of the ulna and there is no visible bridging of the deficiency of bones in the mid-shaft region. A bone graft is now being contemplated.

DISCUSSION

In the past the complications of washing machine wringer injuries in the pediatric age group has been felt to be of little, if any, importance. Our survey would indicate that such a view is in need of modification. It is true that most of these patients escape with little damage. However, in a small percentage of cases serious complications may accompany such injuries. It is mandatory for medico-legal reasons that all of these patients be subjected to study by x-ray examination of the injured part. Fractures are uncommon, but do occur at sufficiently frequent intervals to warrant such investigation in all cases.

It is apparent that soft tissue damage is the complication which occurs with the most frequency in this group of patients. Indeed, this damage may be so extensive and involve so much tissue that surgical procedures, e.g., skin grafts, may be necessary. The degree of damage to the soft tissues

appears to be directly porportional to the length of time that the extremity is caught in the wringer. The most serious damage in this series of cases occurred in the patient whose arm was entrapped in a wringer for an indeterminable length of time. The extent of soft tissue involvement and the course which such involvement will run may not be readily apparent in the early stages. Most of these patients will develop only a moderate swelling of the injured extremity, and a few will progress to abscess formation. Those patients who have obvious soft tissue damage as evidenced by local edema should be followed closely and at frequent intervals for further involvement. The small group of patients who demonstrate extensive necrosis will ultimately have to be subjected to some sort of skin grafting procedure. Early physiotherapy is necessary in this group to prevent the formation of contractures. When skin grafting becomes necessary the period of hospitalization is a long one. These patients, therefore, become economic burdens on their parents and society.

The necrosis that occurs in a small number of these patients is an excellent site for bacterial contamination. In a rare instance, e.g., as in the case reported, bacterial invasion will extend to the underlying osteal parts and produce a local osteomyelitis. It is recommended, therefore, that patients who have localized areas of necrosis be placed on massive antibiotic therapy to prevent such a complication from occurring.

SUMMARY

A group of children who sustained washing machine wringer injuries has been presented. This group represents forty-three patients seen during the last two year period.

The complications that may accompany washing machine wringer injuries have been stressed. In the frequency of occurrence, these include: soft tissue damage, fractures, and localized osteomyelitis.

A representative case of the most serious sequelae which may accompany a wringer injury has been summarized.

A method for the management of these patients has been recommended and the concept that such injuries are of little importance is felt to be in need of modification.

TREATMENT OF PORTAL HYPERTENSION BY PORTAL SYSTEMIC VENOUS SHUNTS

Case Report No. 189

Marshall C. Sanford, M. D.
John A. Washington, M. D.
William L. Sager, M. D.
William W. Orr, M. D.

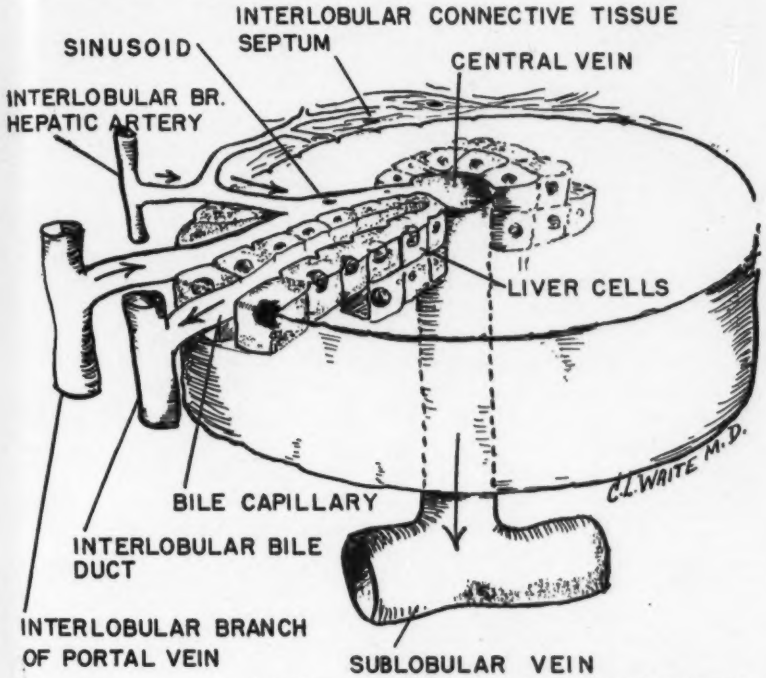
In discussing the subject of portal hypertension it is well to review the anatomy of the portal venous system which drains the intestines, stomach, pancreas, gall bladder, and spleen. It is interposed between two capillary beds and the normal venous pressure is higher in this circuit than in the systemic veins. The microscopic anatomy of the disposition of the terminal branches of the portal vein is shown schematically in Figure 1. The intralobular branches terminate in the hepatic sinusoids which are separated from the bile capillaries by a single layer of hepatic cells. These cells constitute an unloading and loading platform for food and other substances carried by the blood as it passes on into the central vein of the lobule and so on into the hepatic veins which empty into the vena cava. As the portal vein contains little oxygen, arterial blood brought in by the hepatic artery nourishes the liver.

Obstructions of the portal circulation have been classified as follows:

I. Intra-hepatic
   A. Portal cirrhosis (Laennec type)
      1. With cavernomatous transformation of the portal vein
      2. Without cavernomatous transformation of the portal vein
   B. Thrombosis of the hepatic veins
II. Extra-hepatic (Banti's syndrome)
   A. Congenital—obliteration of the portal vein with cavernomatous transformation
   B. Acquired—thrombosis of portal vein or its tributaries
      1. Infectious
      2. Traumatic
      3. Spontaneous
III. Combined type
   Portal cirrhosis with portal vein thrombosis

Portal hypertension can result from an intra-hepatic or an extra-hepatic portal bed block. Those patients with cirrhosis or other types of intrahepatic blockage usually show abnormal liver function tests in addition to splenomegaly, gastro-intestinal bleeding, anemia, and thrombocytopenia. Patients with extra-hepatic portal bed blockage show evidence of portal

hypertension but the liver function tests are normal. Often, in the extrahepatic group, there is a history of upper abdominal trauma or severe infection. In children, portal vein occlusion may result from a continuation of the obliterative processes which normally occur in the umbilical vein and the ductus venosus.



Redrawn from *Physiological Basis of Medical Practice* by Best and Taylor, 3rd. Edition.

FIG. 1. Diagram of Hepatic Circulation

The effect of portal hypertension on the spleen is to produce congestion and chronic hypersplenism. Circulating red blood cells and platelets frequently are reduced, and a tendency to bruise easily may appear. Bone marrow smears show normal or increased production of these elements so that overdestruction by the diseased spleen must be assumed.

With blockage of the normal portal blood flow, new channels must be used to return the blood into the systemic venous circulation. Normally, there are a number of collateral venous pathways through which blood may flow from the portal circuit back into the systemic circuit, the most im-

portant of these being the communications between the coronary veins of the stomach and the lower esophageal veins. These esophageal veins do not anastomose freely with the systemic system so that, frequently, they become greatly enlarged and varicosed. Rupture of these varices accounts for most of the deaths in these patients.

Until rather recently, the surgical procedures have consisted of splenectomy combined with the following attempts to control the secondary manifestations of the portal hypertension, namely, gastro-intestinal bleeding and the formation of ascites.

I. The acute episodes of bleeding—usually from ruptured esophageal varices have been treated by:
   A. Packing the lower esophagus with various hemostatic agents such as gelfoam or oxycel.
   B. Dripping liquid thrombin into the esophagus to speed up clotting.
   C. Esophagoscopy and injection of the varices with sclerosing solutions such as sodium morrhuate.
   D. The introduction of inflatable balloons into the esophagus to compress the varices. Recently a triple lumen, double balloon tube has been devised to accomplish this. It can be passed into the stomach and the lower balloon inflated and pulled up against the cardia. Then the upper sausage-shaped balloon is inflated and the pressure regulated to stop the bleeding. The third lumen is used for aspiration of the stomach to verify cessation of bleeding or may be utilized for feeding if the tube must be left in place for a considerable period of time.

II. Ligation of veins about the lower esophagus and upper stomach has been attempted but without lasting benefit.

III. Resection of the lower esophagus and upper stomach is effective in the controlling of bleeding but is a drastic undertaking in an extremely sick patient.

IV. Peri-esophageal packing with gauze, which is left in place about forty-eight hours and then withdrawn, has been recommended by Garlock in those cases in which it was not possible to perform a venous portal systemic shunt. It is hoped that the granulations thus produced will create sufficient collateral pathways to relieve the tendency of the varices to rupture.

V. In the treatment of ascites, both omentopexy to create new vascular pathways or the introduction of Murphy type buttons through the fascia and muscle into the peritoneum to aid in the more rapid absorption of the fluid, have met with only temporary improvement.

VI. Splenectomy has been the most satisfactory single procedure heretofore employed. Its benefits result from relief of the little under-

stood condition called "hypersplenism" and, more important, by greatly reducing the portal blood flow by probably as much as 40 per cent. The load on the venous collateral pathways is thereby reduced.

A realization of the high mortality and morbidity due to the bleeding from esophageal varices in patients with portal hypertension has stimulated workers to devise means of reducing this high venous pressure by the formation of various types of portal systemic venous shunts. In 1877, Eck demonstrated in experimental animals that blood could be shunted from the portal circulation into the systemic venous circuit by anastomosing the portal vein to the inferior vena cava. This operation was developed so that he might carry out studies on the liver and its relation to metabolism. He suggested that this fistula might be used in cases of portal vein obstruction. Since that time, the procedure was carried out in a few patients, but because of the high mortality associated with the operation its use has been discouraged.

With the introduction of non-suture technique for vascular anastomoses by Blakemore and Lord in 1945, interest in porta-caval shunts was revived. Although the suture technique has now replaced the vitallium tubes, the work they started has continued. The rationale of the procedure is to decompress the hypertensive portal circuit, regardless of the type of obstruction in the portal vein, by producing a shunt between the main portal or one of its tributaries at the time of the splenectomy. In the earlier cases the left kidney was sacrificed and an anastomosis performed between the ends of the splenic and the left renal vein. More recently Blalock and Johns showed that end to side anastomoses are more likely to remain patent than the end to end type so that now the kidney may be preserved.

The technical aspects of these procedures will not be discussed here but it should be mentioned that during operation it is extremely important to only partially, occlude the vena cava and numerous clamps have been devised to accomplish this. Blalock has the impression that "(1) anastomoses of the portal vein and inferior vena cava is preferable, when indicated, to a spleno-renal union in that it will conduct more blood and is more apt to remain patent; (2) if a spleno-renal anastomosis is performed, suture of the proximal end of the splenic vein to the side of the renal vein is preferable to an end to end anastomosis; (3) suture of the divided distal end of the portal vein to the side of the inferior vena-cava is preferable to a side to side anastomosis since the opening is more apt to remain patent."

This work is still in the experimental stages but the procedure appears sound and the results in the treatment of gastro-intestinal bleeding and ascites seem encouraging. Whipple and Blakemore, Blalock, and Linton all report good results in properly selected cases in which it has been possible to perform the operative procedure satisfactorily.

RECOMMENDED PROCEDURE FOR HANDLING PATIENTS WHO ARE BLEEDING FROM ESOPHAGEAL VARICES DUE TO PORTAL HYPERTENSION

I. Control acute bleeding by passage of triple lumen, double balloon tube to compress the varices.
II. Regulate esophageal balloon pressure to the lowest level which controls hemorrhage and verify cessation of bleeding by frequent gastric aspirations.
III. Administer whole blood and vitamin K in large amounts.
IV. After acute bleeding has stopped, demonstrate varices by thick barium swallow or direct esophagoscopy.
V. Feed high carbohydrate diet by mouth, tube, or vein.
VI. Splenectomy combined with portal-systemic venous shunt.

CASE REPORTS

Case No. 1

G. H., a six year old white male, was first hospitalized at the age of nine months because of failure to gain weight. He was born prematurely at seven and one-half months. Early growth and development were normal. An anemia was present, the hemoglobin level being 3 grams, red blood cells 1,800,000. Marked improvement followed two whole blood transfusions. The hemoglobin level rose to 8.7 grams, red blood cells to 3,200,000, and the patient was discharged as improved with the diagnosis of a severe deficiency-type anemia.

Three months later, he was again admitted to the hospital, this time because of difficulty in sitting up. There was no history or evidence of bleeding but once more an anemia was present, the hemoglobin being 3 grams, red blood cells 1,700,000. Following two whole blood transfusions, the patient was considerably improved and allowed to go home. On this admission it was noted that his spleen was considerably enlarged. During frequent medical check ups made in the ensuing year it was noted that the patient tended to bleed easily from the slightest cut. Vitamin K, liver extract, and iron administration seemed to remedy this difficulty.

At four years of age he was again admitted to Children's Hospital, this time because of sudden tiredness and fever of forty-eight hours' duration. Severe tonsillitis accounted for the fever. The spleen was found to have reached enormous proportions extending down to the iliac crest. A tentative diagnosis of congestive splenomegaly or Banti's syndrome was made. Soon after admission, tarry stools were noted, followed by the vomiting of bright red blood with some clots. The melena and hematemesis continued for several days requiring an almost continuous transfusion of whole blood, but the bleeding finally stopped spontaneously. Barium studies of the esophagus were made but they failed to reveal esophageal varices possibly because too thin a mixture was used.

A splenectomy was performed on July 14, 1947. The pathological report confirmed the diagnosis of Banti's syndrome. The post-operative course was uneventful and he was discharged to be followed at home.

A few months later he was admitted to the hospital because of epistaxis and fever of four days' duration. He developed chicken-pox a few days later. The anemia was present again. The hemoglobin was 6 grams and the red blood cells 2,100,000. Improvement followed two whole blood transfusions and on discharge his hemoglobin had risen to 11 grams and the red blood cells to 3,700,000.

The next admission was on February 16, 1950 when the patient was six years old. That day he had vomited two cupsfull of bright red blood and had passed several tarry stools. The hemoglobin was 7 grams and the red blood cells were 2,700,000. Barium visualization of the esophagus revealed large varices in the lower portion. He was given blood transfusions and discharged.

Two months later, he was re-admitted for evaluation of his liver function prior to a porta-caval shunt to compress the hypertensive portal circuit. Physical examination was unchanged. The hemoglobin was 10 grams and the red blood cells were 3,500,000. The white count was 6,000 and the platelets numbered 50,000. Thymol turbidity was 0.5 units and the prothrombin activity was 75 per cent in thirteen seconds. The cephalin flocculation determination was negative after twenty-four and forty-eight hours. The patient was given Vitamin K and whole blood and maintained on a high carbohydrate diet.

On April 29, 1950, through a right combined thoraco-abdominal approach, an anastomosis was performed between the distal end of the divided portal vein and the side of the inferior vena cava. The patient's post-operative course was uneventful. Barium study of the esophagus twelve days following the operation failed to reveal the varices. Since discharge, he has been completely asymptomatic and although his activity has been unrestricted, he has no recurrence of his bleeding.

Case No. 2

R. S., a nineteen year old white male was first seen in Children's Hospital in August of 1936 at the age of six years because of abdominal enlargement and failure to gain weight during the preceding year. Birth and development had been normal and there was no history of severe infection or abdominal trauma. Physical examination revealed a thin, fairly well-developed boy whose spleen was considerably enlarged. The left upper quadrant was completely filled with a non-tender mass which extended to the mid-line and its lower margin was at the level of the umbilicus. The liver was not enlarged. Laboratory studies including liver function tests were normal, except for an anemia with a hemoglobin of 9.5 grams and red blood cells

3,500,000. Several blood transfusions resulted in improvement and the patient was discharged.

In November of 1937, at the age of seven, the patient was readmitted because of vomiting large quantities of blood. There was no history of trauma and the patient's health had been good during the preceding year. Physical examination revealed a thin, extremely pale, acutely ill child whose physical status had changed little since the last examination, except that the spleen appeared to have enlarged, and now the lower border reached the left iliac crest. Liver function tests were again normal; hemoglobin was 8 grams; red blood cells numbered 2,800,000, and platelets were 43,000. The diagnosis of splenic anemia with bleeding from esophageal varices was made and the patient was treated with transfusions of whole blood.

On December 21, 1937 a splenectomy was performed and pathological sections confirmed the diagnosis of Banti's syndrome. His recovery was uneventful and he was discharged as improved, and with no evidence of further bleeding.

Following discharge from the hospital, he continued to do well. He was able to go to school, but did not participate in athletics.

On May 9, 1950 he began having massive hematemeses, vomiting as much as 500 cubic centimeters of bright red blood at a time. He was rushed to Emergency Hospital where whole blood transfusions were started immediately. Physical examination revealed an acutely ill, extremely pale, nineteen year old white boy who was extremely apprenhensive, but in no apparent distress. His liver was palpable at the costal margin. The abdomen was slightly distended. The bleeding continued to a lesser degree for several days associated with melena. In order to control the bleeding a modified Miller-Abbott tube was passed into the stomach, inflated, and pulled up to compress the varices. This device controlled the bleeding fairly well, but was later replaced with a triple lumen double balloon Sengstaken esophageal tube. This was passed into the stomach and the lower balloon inflated and pulled up against the cardia. The proximal sausage-shaped balloon was then inflated and the pressure regulated to the lowest level which would sufficiently compress the varices to control the bleeding. This was verified by aspiration of the lumen which extended into the stomach. This tube was left in place for forty-eight hours and then removed. Barium studies of the esophagus demonstrated varices, but soon after the completion of the studies, hematemesis recurred. The compression tube was reinserted and the patient fed a high carbohydrate liquid diet through the tube. Liver function tests were normal. The blood transfusions were continued.

On May 19, 1950, through a right combined thoraco-abdominal approach, a side to side anastomosis was performed between the portal vein and the

inferior vena cava. The portal vein was considerably scarred and somewhat cavernomatous. Numerous collateral vessels ran along the main portal trunk, but a satisfactory union between the two vessels was performed. The patient made an uneventful recovery from this operation and was up and about within a week. Nineteen transfusions were required in the period before and during operation. The esophageal compression tube was removed seventy-two hours after the operation and the liquids which he had been receiving through the tube were then given by mouth. On the fifth day, a soft diet was begun and later a regular diet was allowed.

On June 6, 1950, the patient was discharged from the hospital considerably improved. Since that time, he has gotten along well and has had no recurrence of his bleeding. His activity has been restricted somewhat but he was allowed to go to the beach, where he is enjoying himself at the present writing.

SUMMARY

1. The medical aspects of portal hypertension and the types of portal bed block are discussed.

2. Two cases of gastro-intestinal bleeding from ruptured esophageal varices which have been treated by portal systemic venous shunt are reported.

3. The usual methods of treating cases of this type are discussed.

4. Recommended procedures for handling these cases are outlined.

5. The production of a portal systemic venous shunt appears to be a sound, surgical procedure for the treatment of selected cases of gastro-intestinal bleeding or ascites associated with portal hypertension.

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APLASIA OF THE LUNG WITH PSEUDO-BIFURCATION OF THE TRACHEA

Case Report No. 190

Adrian Recinos, Jr., M. D.
Charles L. Waite, M.D.

L. H. 48-3447

Aplasia of the lung is an uncommon entity. A survey of the literature reveals forty-five authentic cases.[2, 7, 8, 9] The increasing frequency with which aplasia is being recognized suggests that it is not so rare as was previously considered. In the past decade congenital absence of a lung has been recognized prior to death on several occasions.[6, 7, 8, 9] A positive diagnosis during life is based on bronchographic and bronchoscopic observations which show, instead of a normally bifurcating trachea, the absence or blind vestige of one of the main stem bronchi.

Aplasia of the lung is not incompatible with longevity. It has been discovered as an incidental finding at autopsy in adults dying of other causes.[1, 4, 5] Garber's[8] case, a four year old boy, was in good health several years after the diagnosis was made.

The following report is intended to add another case to the growing number of pulmonary aplasias and to point out a potential difficulty—in this case virtually an impossibility—in making a diagnosis ante mortem.

CASE REPORT

L. H., a seven week old white male, was admitted to the hospital because of several episodes of cyanosis on the day of hospitalization associated with cold, cough, and choking of three days' duration. He had had another cold at the age of three weeks and his mother stated that he had always "gagged on mucous."

The patient was the product of his mother's sixth pregnancy. Labor was induced in the ninth month because of hypertension. The baby was delivered uneventfully after a two hour labor and weighed six pounds, twelve ounces at birth. He became jaundiced on the third day of life and remained icteric until two weeks of age. At this time he was taken home from the hospital, weighing only five pounds, ten ounces. The patient was breast fed and was given no other nourishment. The father, mother, and four siblings were living and well at the time of his admission to the hospital. One sibling, however, died ten hours after birth from undetermined causes.

Examination on admission revealed a small, undernourished infant in acute respiratory distress. The temperature was 99.2 F. The skin was cold and clammy and there was cyanosis about the mouth and in the nail beds. Respirations were noisy and labored but there was no retraction of the chest

or diaphragm. The fontanel was widely patent but not bulging. There was considerable mucous in the throat. The heart was rapid but the sounds could not be heard clearly because coarse, moist râles monopolized the auscultation over both lung fields.

A blood count disclosed a hemoglobin of 12 grams; 3,200,000 erythrocytes; 14,200 leukocytes with 60 lymphocytes, 38 neutrophiles, 1 monocyte, and 1 basophile per 100 cells. The urinalysis was normal. A throat culture yielded non-hemolytic *Staphylococcus albus* and pneumococcus.

An x-ray of the chest was interpreted as showing atelectasis of the left lung (Figure 1). The mediastinum was shifted to the left, the diaphragm was

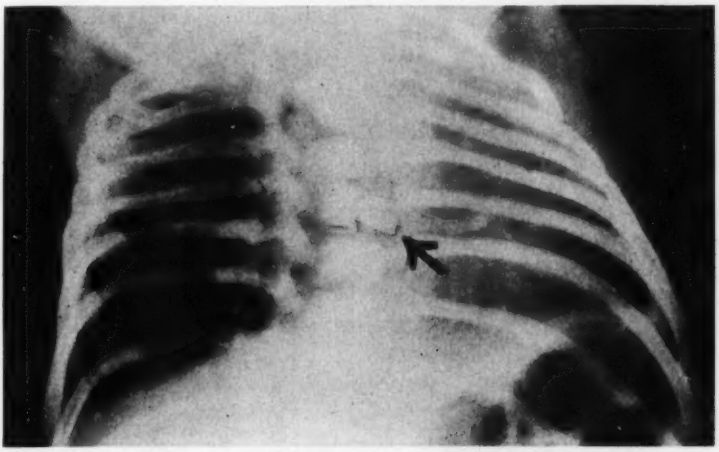

FIG. 1. L. H.: AP view of the chest showing thoracic hemivertebra and x-ray signs of atelectasis on the left side.

eventrated, and the intercostal spaces were narrowed on that side. In addition, there was a scoliosis with an abnormal seventh thoracic vertebrae at the apex of the curve. An azygos lobe was believed present.

The patient was placed in an oxygen tent and his throat was suctioned frequently. Penicillin was given intramuscularly and parenteral fluids were administered. In spite of these measures the baby failed to improve and continued to have dyspnea, intermittent cyanosis, and accumulation of tenacious secretions in the throat. Repeat x-ray on the fifth day showed no essential change. The course was progressively downhill and the baby died on the twelfth hospital day.

PATHOLOGICAL FINDINGS

Post-mortem examination revealed a tiny, malnourished infant appearing much younger than the stated age. The lips and finger nails were cyanotic.

The skin was thin in texture and "dusky" in color and there was a paucity of subcutaneous fat.

The sternum was removed and the thorax opened (Figure 2). The heart was noted to be shifted slightly to the left but apparently not enlarged. There was no free fluid. No pulmonary tissue was seen in the left chest. In its place there was a mass of loose connective tissue medially and in-

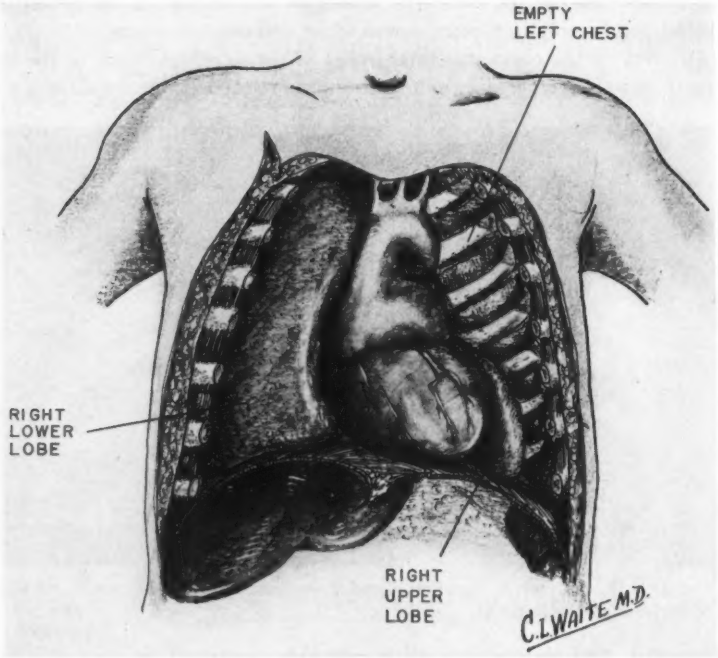

FIG. 2

feriorly and elsewhere a cavity lined by parietal pleura and the inner rib cage. Dissection of the connective tissue mass revealed it to cover and enclose a quadrangular portion of lung tissue measuring 3 by 2 centimeters and partly covered by the heart. The connective tissue "envelope" was 2 to 3 millimeters thick and was adherent to the diaphragm and to the lateral parietal pleura.

The right thorax contained no fluid or adhesions and it was filled by a dark rose-colored crepitant "lung" which was not differentiated into lobes and whose hilum could be neither visualized nor palpated.

A block dissection of the entire respiratory tract and the heart and great

vessels was performed. The trachea was grossly normal in appearance and caliber. It contained a tenacious mucoid material which extended into the large bronchi. The trachea divided into the vicinity of the fifth thoracic vertebra into two branches of equal size simulating a true "bifuraction." The left branch entered the small block of lung in the left thorax without subdividing. It was crossed *posteriorly* by a branch of the RIGHT pulmonary artery. The right branch of the trachea gave off a bronchus medially to an atelectatic portion of lung in the mediastium behind the heart and proceeded to the large, crepitant pulmonary mass filling the right chest. A "bronchial bud," a small, solid, slightly-elevated tracheal mass measuring 4 to 5 millimeters in diameter, was present on the right side of the trachea 0.5 centimeters above the "bifurcation."

The entire pulmonary tissue consisted of three continuous segments divided partly by fissures, resembling three lobes of a single right lung. The largest segment (right) filled the right thorax; the middle segment lay behind the heart; and the left segment was in the inferior medial portion of the left thorax. The right and middle segments were supplied by the right-sided division of the trachea; the left by the left division.

The large, crepitant unilobar mass in the right thorax was sectioned and noted to contain moderate congestion and edema on a well aerated parenchyma. Microscopic examination revealed a preponderance of normal-sized or dilated alveoli, some containing exfoliated cells and macrophages. There was considerable congestion and edema. The middle segment, smallest of the three, measured 2 by 1.5 by 1.5 centimeters. It was dark and totally airless. The left segment was darker than the right and lighter than the middle. On gross section, it appeared atelectatic and congested. Histologic examination of the middle and left lung segments showed a uniform and marked atelectasis.

The pericardium appeared normal. The heart was normal in weight and size and was shifted slightly to the left. The aorta and the aortic arch appeared normal and the ductus arteriosus was closed.

The left pulmonary artery was entirely absent. The right pulmonary artery branched 1.5 centimeters from its origin, the lower branch entering the unilobar "lung" in the right chest. The upper branch was traced superiorly and posteriorly for a distance of 1.5 centimeters and then turned sharply to the left partly encircling the right "main" bronchus and proceeding posteriorly to it. It then crossed the midline anteriorly to the esophagus, crossed the bronchus of the left lobar mass posteriorly, and entered that portion of the lung. The myocardium, chambers, and valves of the heart were quite normal. The foramen ovale was patent, measuring 3 millimeters in diameter.

Removal of the heart and right lung revealed a marked left dorsal scolio-

sis with the apex formed by a seventh thoracic left hemivertebra. No rib was attached to this abnormal vertebra. The left posterior thoracic cavity was consequently smaller than the right.

The only other abnormality was found in the urinary tract. The right kidney and ureter and the right renal vessels were totally absent, the right adrenal lying freely in the upper lumbar gutter. On the left there was a moderate hydronephrosis and hydroureter with stenosis of the ureter at the junction of the middle and upper thirds.

Interpretation of Anatomical Findings:

There seems little doubt that this was a case of absence of the left lung with the complex picture at death resulting from a turning, twisting migration of the lobes of the right lung toward the left pleural cavity. The right upper and middle lobes have migrated to the left, the upper lobe entering the left chest and the middle lobe reaching the mediastinum where it has become lodged and compressed behind the heart. The right upper lobe reached the left chest by moving posteriorly and to the left (Figures 3, 4, 5) pulling the middle lobe and the right pulmonary artery with it and rotating the trachea 180 degrees clockwise as viewed from above (Figure 7). In the course of this rotation the bronchial bud moved from the left to the right side of the trachea.

The right upper lobe bronchus is known as the "eparterial bronchus" because it is normally crossed *anteriorly* by the right pulmonary artery. In our case the bronchus was crossed *posteriorly* because it has moved posteriorly and to the left and rotated 180 degrees on the trachea (viewed from above) pulling the artery after it (Figure 7). The right lower lobe has expanded in all directions (Figure 6) filling the entire right pleural cavity and assuming the entire respiratory load.

The apparent bifurcation of the trachea actually represents the division of the right main stem bronchus into upper and lower branches. The fact that the three segments of the lung are contiguous, that they are all supplied by the right pulmonary artery, and that the left pulmonary artery is absent gives further evidence that the upper, middle, and lower lobes of the right lung account for all the pulmonary tissue present in this case and that the left lung is absent. The unilateral absence of another paired organ (in this case the right kidney) and the presence of a hemivertebra[10] lend further support to the diagnosis.

The pathologic diagnosis in this case was:

Aplasia of the left lung and the left bronchus, congenital with
Herniation of the right upper lobe of lung into the left thoracic cavity and right middle lobe into the posterior mediastinum.
Pulmonary atelectasis, right upper and middle lobes.
Absence of the left pulmonary artery, congenital.

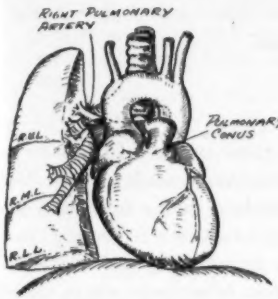

FIG. 3

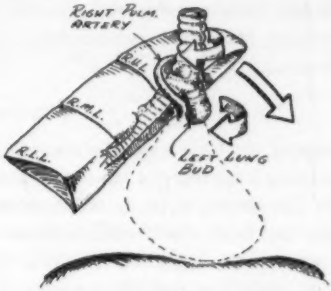

FIG. 4

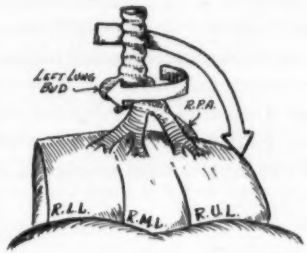

FIG. 5

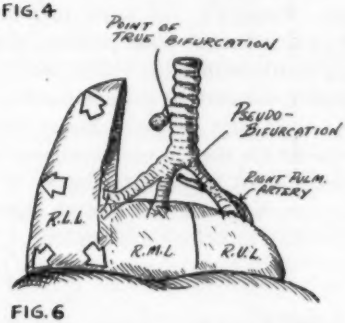

FIG. 6

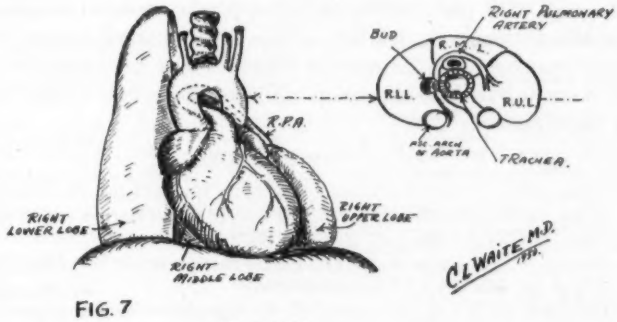

FIG. 7

FIGS. 3-7.

Patent foramen ovale.

Passive congestion of the spleen and accessory spleen.

Absence of the right kidney, ureter, renal artery and vein, congenital.

Hydronephrosis and hydroureter, left, due to congenital stenosis of the ureter at the junction of the upper and middle thirds.

Dorsal scoliosis, due to
Seventh thoracic hemivertebra, congenital.
Accessory spleen.

DISCUSSION

Aplasia of the lung is usually classified into three types.[6] In true or total aplasia there is no trace of the lung, bronchus or blood vessels on the affected side. In the second type, a rudimentary bronchus, in the form of a small outpocketing from the trachea with no sign of lung tissue is present. In the third type, more accurately described as a hypoplasia, the bronchus is small and ends in a vestigial lung without lobes. Most cases are of the first type. Ordinarily, the heart and mediastinum are displaced toward the affected side and some portion of the hyperplastic and emphysematous lung herniates into the contralateral thoracic cavity. Caffey[10] has noted a frequent association of pulmonary aplasia and hemivertebrae and suggests that they may be two components of a congenital syndrome. Our case certainly fits the picture. A variety of other congenital anomalies has been described. Of particular interest to us was Killingsworth's case[5] which, like ours, had in addition to an absence of one lung, a total absence of the contralateral kidney and ureter.

SUMMARY

A case of congenital aplasia of the left lung in a nine week old infant is presented. This case, unlike those previously reported, simulated one of true bifurcation of the trachea, a false division being formed by the right lower bronchus and the displaced right upper bronchus. Bronchoscopic and bronchographic examination would not have been of help in establishing the diagnosis.

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21st ANNUAL SCIENTIFIC ASSEMBLY OF
THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
HOTEL STATLER, OCTOBER 2, 3, AND 4, 1950

Subjects	*Sponsers*
ACTH and Cortisone in Rheumatic Fever and Related Conditions	F. R. Leyva, M.D.; M. P. Schultz, M.D.; B. J. Walsh, M.D., and E. C. Rice, M.D., Children's Hospital.
Congenital Hernias	Philip A. Caulfield, M.D.; Gerald McAteer, M.D., and Robert Pender, M.D., Children's Hospital.
Neonatal Morbidity and Mortality at the Children's Hospital, Washington, D. C.	J. P. McGovern, M.D. and W. L. Sager, M.D., Children's Hospital.
Urogenital Anomalies and Pediatric Mortality	J. M. LoPresti, M.D. and C. L. Waite, M.D., Children's Hospital.
Diseases of the Ear, Nose and Throat	Irving Hantmann, M.D., Dept. of Otolaryngology, Georgetown Medical School.
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Transorbital Lobotomy for the Relief of Intractable Pain	Walter Freeman, M.D. and Jonathan Williams, M.D., George Washington Medical School.
Bronchiectasis Secondary to Tuberculosis	Daniel L. Finucane, M.D., Glen Dale Sanitorium, Washington, D.C.
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Tantalum Mesh in Repair of Hernias	E. A. Cafritz, M.D.
Surgery of Gastric Lesions	William W. Chase, M.D. and William E. Ferguson, M.D.
The Surgical Anatomy of the Peritoneal Fossae	Othmar Solnizky, M.D. and Philip Burka, M.D., Georgetown Medical School.
Intra-arterial Cancer Chemotherapy	Calvin T. Klopp, M.D., Dept. of Surgery, George Washington Medical School.
Parenteral Carbohydrate Therapy—Use of Invert Sugar	Jacob J. Weinstein, M.D., George Washington Medical School and Gallinger Municipal Hospital.
Cephalopelvimetry	Fred O. Coe, M.D., of Groover, Christie and Merritt.
Extraperitoneal Cesarean Section	Thomas M. Leonard, M.D.; William T. Lady, M.D., and Major Humbert Riva, M.C., Walter Reed Army Hospital.
Original Tocographic Studies Upon the Parturient Uterus	Samuel M. Dodek, M.D., George Washington Medical School
Department of Surgery—Doctors Hospital	Charles S. White, White, Wilson, and White.
A Study in Gross Osteopathology	William J. Tobin, M.D. and D. J. O'Regaan, M.D., Georgetown Medical School, and T. D. Steward, M.D., National Museum, Smithsonian Institution.
Flat Feet in Young Children	Maurice Herzmark, M.D.
Functions of a Blood Grouping Laboratory	John B. Ross, M.D. and Oscar B. Hunter, Jr., M.D., Blood Grouping Laboratory, Wash., D.C.
Neurological Causes of Neck, Shoulder, and Arm Pain	Arthur A. Morris, M.D., Georgetown Medical School
Tumors of the Thyroid Gland	William L. Amoroso, Jr., M.D. and Robert J. Coffey, M.D., Depts. of Surgery and Pathology, Georgetown Univ. Medical Center.
Trinder Tampon for the Control of Intractable Nasal Hemorrhages	John J. Trinder, M.D., Georgetown Medical School.

CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: William M. Crowell, M.D.
Francis J. Troendle, M.D.
By invitation: Richard Jackson, M.D.

William M. Crowell, M.D.

R. M., a six year old colored male, was admitted to this hospital with the chief complaint of "pain in the stomach" and the story of having been well until twenty-four hours prior to admission. At this time he awoke from his sleep, vomited, and complained of a stomach ache. He reawoke several times during the night crying. On the following day, the day of admission, he vomited three times and still complained of abdominal pain. One bowel movement was normal and all food was refused after breakfast. The physical findings on admission were those of a well developed and well nourished colored boy who was cooperative. The temperature was 99.0° F., the pulse 88, and the respirations 22. The abdominal findings were a soft to rigid abdomen with questionable right lower quadrant tenderness and guarding when awake which disappeared when asleep. A rectal examination was negative. Laboratory studies revealed 8,200 leukocytes with 68 per cent segmenters and 2 per cent bands, normal erythrocytes and hemoglobin, and a negative urinalysis. By this time it was 11:00 P.M. so it was decided to wait until morning for repeat laboratory studies and reevaluation.

The past history was non-contributory as was the family history. No history of a similar episode could be elicited.

The following morning, about twelve hours after admission, laboratory studies revealed a negative sickling preparation, 7,200 leukocytes with 74 per cent segmenters and 6 per cent bands, slightly decreased erythrocytes and hemoglobin, and a negative urinalysis. Physical examination was negative but in view of the preceding history, a routine appendectomy was performed. The appendix was described as being "acutely inflamed throughout and bound down by numerous adhesions." A "moderate amount of clear straw-colored fluid was found in the abdominal cavity." The immediate post-operative condition was good.

For three days post-operatively he did well and behaved normally; however, on the fourth and fifth days he complained of abdominal pain but continued to eat and was afebrile. On the morning of the fifth day the sutures were removed from the abdominal incision, and the resident called the wound "satisfactory." That afternoon the abdomen became distended and involuntarily rigid. He vomited a large amount; peristalsis was not heard, and the temperature was 105.8° F.; the patient was vomiting brown

non-fecal-smelling material and the abdomen was still distended. The chest was clear at the time and the blood pressure was 100/70. A hemogram at 11:00 P.M. revealed 6,700 leukocytes with 50 per cent segmenters and 46 per cent lymphocytes. The hemoglobin was 13.5 grams with 5.8 million erythrocytes. A film of the upright abdomen revealed "several small bowel fluid levels." At midnight fluids were started to combat shock, dehydration, and continuous Wagensteen suction. A medical consultation suggested chemotherapy and biotherapy, which was begun in adequate amounts. The incision was explored and no evidence of infection was found. Cyanosis appeared intermittently but was controlled by the shock therapy. By morning, the sixth post-operative day, the temperature was 104° F., the pulse was feeble, the blood pressure was unobtainable and the same supportive fluid therapy, chemotherapy and biotherapy were being employed. The abdomen remained distended but was decreasing; however, palpation remained painful. The diaphragm was high and peristalsis was absent. The leukocyte count was 8,600 with 36 per cent neutrophiles and 64 per cent lymphocytes. An exploratory laporatomy was considered but the patient expired at noon.

DISCUSSION

Richard Jackson, M.D.

This six year old colored male was admitted to Children's Hospital complaining of abdominal pain of two days' duration with nausea and vomiting. On physical examination the signs were variable, but could be due to one of several surgical conditions of the abdomen. By far the most common condition resulting in an acute abdomen in this age group in is appendicitis. Even though this child was operated on for appendicitis, I think we should consider two other conditions that are not uncommon in this age group. The first is intussusception and the second is volvulus. The history as previously given does not fit in with intussusception. In intussusception we almost always have severe cramp-like pains that usually will cause the patient to cry out. The absence of a palpable mass and current jelly material makes me feel that we do not have to consider intussusception in this case. Volvulus, however, presents an entirely different picture and it is not uncommon to find that volvulus will give only mild recurrent abdominal pain, and the history in this case is quite suggestive of a recurrent type of mild abdominal pain. The physical findings also might fit in with volvulus because the abdominal findings were not constant, there being no area of localization of tenderness as one would expect in acute appendicitis.

There are other medical conditions which may give a six year old patient the signs and symptoms or an acute abdomen such as diabetes, inflamma-

tory diseases involving the kidney, mesenteric lymphadenitis secondary to upper respiratory infection. However, with the information presented in this case, I think we are safe in ruling out any strictly medical condition that might give signs and symptoms of an acute abdomen. The temperature of 99 might be found in an early intestinal obstruction or in appendicitis.

The second part of this case is most interesting in that apparently the patient made an uneventful recovery from the operation until the fourth postoperative day, at which time the patient complained of a return of the abdominal pain, associated with a high fever, distention, muscle rigidity over the entire abdomen, shock, and death. Any child this age with a surgical condition of the abdomen that might lead to such a termination as this is quite rare. I think the three most common conditions that we might consider are: (1) a rupture of an abdominal abscess which rapidly contaminates the entire peritoneal cavity producing severe peritonitis associated with shock, and which may end up in a fatality; (2) a complete blowout of the appendiceal stump spilling liquid fecal material over the entire abdominal cavity. This practically always results in death; (3) intestinal obstruction which has been allowed to advance to gangrene of the bowel with resulting peritonitis, shock, and a fatal termination.

I think in this case that it is almost impossible to differentiate between these three conditions. I must, however, make a definite diagnosis in order to make the conference more interesting. I will say that the patient had intestinal obstruction, which was not detected at the first operation and advanced to the stage of gangrene and perforation of the bowel. Regardless which of the three conditions caused the patient's death after the signs of shock had appeared due to peritonitis, it is practically impossible to do anything from a surgical standpoint to offset the natural course of the disease.

PATHOLOGIC DISCUSSION

John E. Cassidy, M.D.

Dr. Jackson has given us a fine and interesting discussion of this case. In his differential diagnosis, he has talked about the surgical causes of the acute abdomen in children. For the sake of completeness, we should mention some of the non-surgical causes. The most common cause of abdominal pain, nausea and vomiting is acute gastritis or gastro-enteritis, usually associated with dietary indiscretion. Some of the other causes are diabetic acidosis, acute glomerulonephritis, acute pyelitis and/or cystitis, and in the colored race, sickle cell crises.

At autopsy, the principal findings were in the peritoneal cavity and the abdominal viscera. The peritoneal cavity contained approximately two liters of purulent, greenish-gray foul smelling liquid material, cultures of which revealed a non-hemolytic escherichia coli. The peritoneum was

edematous and acutely inflamed, the loops of the bowel being plastered together by a fibrino purulent exudate. In the region of the cecum, there was a peri-appendiceal abscess which contained the necrotic appendiceal stump and one cotton ligature. There was a direct communication between the cecum and peritoneal cavity via the appendectomy site.

Other significant findings were a hepatitis, adrenalitis and pancreatitis, which were most likely secondary to an overwhelming infection. There were several ascaris lumbricoides present in the ileum. Hearts blood culture grew out a gram negative bacillus, which could not be identified.

It is evident then that this patient had an appendectomy and an acutely inflamed appendix was removed. His post-operative course was normal until the fifth day at which time the appendiceal stump gave way and fecal contents spilled into the peritoneal cavity producing the picture and sequelae which have been mentioned.

This case brings to the fore the oft heard discussion about whether an appendix stump should or should not be inverted or whether it makes any difference. Probably some of our surgeon friends can bring us up to date on this point.

CAMPAIGN FOR ROUTINE IMMUNIZATIONS IN CHILDREN

The District of Columbia Medical Society on Child Welfare is sponsoring a campaign throughout the month of September aimed at improving the level of immunization in the pediatric population.

With the cooperation of the Bureau of Education, through the medium of newspapers and radio messages, people are urged to check with physicians or health clinics on the immunization status of their children. Physicians are exhorted to cooperate with the campaign and to see that children under their care have their immunizations up to date.

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